U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 03 August 2005

Case Nos. 2004-BLA-5090
2004-BLA-16

In the Matter of:

HELEN E. CLAAR, survivor of and
on behalf of ALDEN H. CLAAR
Claimant

v.

ISLAND CREEK COAL CO.
Employer

and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS
Party-in-Interest

Heath M. Long, Esq.
For the Claimant

Ashley M. Harman, Esq.
For the Employer

Before: DANIEL L. LELAND
Administrative Law Judge

DECISION AND ORDER—AWARDING BENEFITS

This case arises from claims for benefits under the Black Lung Benefits Act, 30 U.S.C. § 901 *et seq*. In accordance with the Act and pertinent regulations, this case was referred to the Office of Administrative Law Judges by the Director, Office of Workers' Compensation Programs for a formal hearing.

Benefits under the Act are awarded to persons who are totally disabled within the meaning of the Act due to pneumoconiosis or to the survivors of persons whose death was caused by pneumoconiosis. Pneumoconiosis is a dust disease of the lung arising from coal mine employment and is commonly known as black lung.

At a formal hearing conducted in Ebensburg, Pennsylvania on February 14, 2005, all parties were afforded a full opportunity to present evidence and argument, as provided in the Act and Regulations found in Title 20, Code of Federal Regulations. Regulation section numbers mentioned in this Decision and Order refer to sections of that Title. At the hearing, Director's exhibits 1-136, Claimant's exhibits 1-2, and Employer's exhibits 1-8 were admitted into evidence. In addition, I granted Claimant permission to depose Dr. Begley. (TR 19). His report was marked and entered into the record as CX 3. The employer filed a post-hearing brief.

ISSUES

The employer stipulated that the miner had nine years of coal mine employment in its employ and that he had simple coal workers' pneumoconiosis arising out of coal mine employment, which establishes a material change in conditions and grounds for modification.[1] The District Director credited the miner with 19.37 years of total coal mine employment, which was not contested by either party and is consistent with the record. (DX 132, 133). Thus, I find that the miner was a coal miner within the meaning of the Act for 19.37 years. Employer does not contest that it is the Responsible Operator. (TR 11). The issues remaining in the miner's claim are whether he was totally disabled and whether his disability was due to pneumoconiosis. The sole issue in the survivor's claim is whether the miner's death was due to pneumoconiosis.

FINDINGS OF FACT AND CONCLUSIONS OF LAW[2]

Procedural History[3]

Claimant, Alden Claar, filed this second claim for benefits on October 27, 2000.[4] (DX 135) The District Director denied benefits by Proposed Decision and Order on December 21, 2000 and April 24, 2001. (DX 37). The claimant disagreed with the determination and requested a formal hearing. By Decision and Order dated July 23, 2002, Administrative Law Judge Michael P. Lesniak denied benefits. (DX 66). The miner died on February 2, 2002, and his widow, Helen E. Claar, requested modification of Judge Lesniak's decision with the District Director on July 23, 2002. (DX 67). The District Director granted the request for modification in a Proposed Decision and Order dated July 10, 2003 and Employer requested a formal hearing with the Office of Administrative Law Judges. (DX 83, 85).

The miner's widow filed a survivor's claim on September 16, 2002. (DX 91). In a Proposed Decision and Order, the District Director awarded benefits on July 10, 2003, and

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[1] *See* Employer's brief at 4.

[2] The following abbreviations have been used in this decision: DX = Director's exhibit; EX = Employer's exhibit; CX = Claimant's exhibit; TR = Transcript of the hearing; BCR = Board-certified radiologist; and B = B reader of x-rays.

[3] Given the filing date of this claim, subsequent to the effective date of the permanent criteria of Part 718 (i.e., March 31, 1980), the regulations set forth at 20 C.F.R. Part 718 will govern its adjudication. Because the miner's last exposure to coal mine dust occurred in Pennsylvania, this claim arises under the jurisdiction of the U.S. Court of Appeals for the Third Circuit. *See Broyles v. Director, OWCP*, 143 F.3d 1348, 21 BLR 2-369 (10th Cir. 1998).

[4] Claimant's first claim for benefits, dated December 31, 1986, was denied by Administrative Law Judge Charles P. Rippey on February 16, 1989. (DX 42).

Employer contested the findings and requested a formal hearing on July 22, 2003. (DX 127, 129). Both claims were referred to the Office of Administrative Law Judges on October 14, 2003 (DX 132, 133).

The findings of fact and conclusions of law that follow are based upon my analysis of the entire record, including all documentary evidence admitted, arguments made, and the testimony presented. Where pertinent, I have made credibility determinations concerning the evidence.

Background

Claimant is the widow of the miner who was born on February 21, 1932 and died on February 2, 2002. (DX 69, 101). The miner's coal mine employment ended in 1980 when the mine shut down. (DX 60 at p. 12). The miner's last coal mine job was as a unitrack operator, which required him to scoop up loose coal and rock dust and dust the top of the roof. (DX 60 at pp. 14-15). This required lifting 50-pound bags on a regular basis. *Id.* The miner also had to lift crossbars weighing 200 pounds. (DX 60 at p. 16). The miner smoked one half to one pack of cigarettes per day starting in the early 1950s until the 1970s. (DX 60 at pp. 17-18).

Modification and Duplicate Claim

Claimant submits that the evidence establishes a material change in conditions pursuant to 20 C.F.R. § 725.309(d) (2000). This section provides that in cases where a claimant files more than one claim and a prior claim has been finally denied, later claims must be denied on the grounds of the prior denial unless the evidence demonstrates "a material change in conditions." The Third Circuit's standard for determining the existence of a material change in conditions provides that an administrative law judge must consider all of the new evidence, both favorable and unfavorable, to determine whether the miner has proven at least one element of entitlement that was previously adjudicated against him. If a claimant establishes the existence of one of these elements, he will have demonstrated a material change in conditions as a matter of law. The administrative law judge must then consider whether all of the evidence of record, including evidence submitted with the prior claim, supports a finding of entitlement to benefits. *LaBelle Processing Co. v. Swarrow*, 72 F.3d 308, 317-8 (3rd Cir. 1995); *See Sharondale Corp. v. Ross*, 42 F.3d 993, 997-98 (6th Cir. 1994).

The regulations further provide that modification of an order may be sought at any time before one year after the denial of the claim. Specifically, the terms of an award or the decision to deny benefits may be reconsidered upon the showing of a "change in conditions" or a "mistake in a determination of fact." 20 C.F.R. § 725.310 (2003). In evaluating a request for modification, it is not enough that the administrative law judge conduct a substantial evidence review of the district director's finding. Rather, the parties are entitled to *de novo* consideration of the issue. *Kovac v. BCNR Mining Corp.*, 14 B.L.R. 1-156 (1990), *aff'd on recon.* 16 B.L.R. 1-71 (1992); *Dingess v. Director*, *OWCP*, 12 B.L.R. 1-141 (1989); *Cooper v. Director, OWCP*, 11 B.L.R. 1-95 (1988). In addition, even if a change in conditions is not established, evidence must be considered to determine whether a mistake in a determination of fact was made, even where no specific mistake of fact was alleged. *See O'Keeffe v. Aerojet-General Shipyards, Inc.*, 404 U.S. 254, 256 (1971); *Jessee v. Director, OWCP*, 5 F.3d 723 (4th Cir. 1993); *Consolidation Coal*

Co. v. Director, OWCP [Worrell], 27 F. 3d 227 (6th Cir. 1994). In this case, however, Claimant alleges that Administrative Law Judge Lesniak made a mistake in fact. (DX 67).

The miner's first claim was finally denied by Judge Rippey on February 16, 1989. (DX 42). Judge Rippey found that the claimant established the existence of pneumoconiosis by virtue of the now defunct "true doubt" rule, and also found that he was not totally disabled due to pneumoconiosis. In this, his current claim for benefits, Judge Lesniak found that Claimant did not establish the presence of pneumoconiosis, but found that he was totally disabled. Thus, in order for Claimant to prove a change in conditions, the new evidence must be evaluated to determine whether any of those elements can now be established.

Employer has stipulated to the presence of simple coal workers' pneumoconiosis, and I find the miner was totally disabled due to pneumoconiosis, which establishes a change in conditions pursuant to § 725.310. In addition, I find that Judge Lesniak did not make a mistake of fact with the evidence that was before him at the time; however, the autopsy evidence reveals that the miner had pneumoconiosis and therefore his opinion should be modified to reflect this.

Medical Evidence

Chest x-rays

As Employer conceded that the miner suffered from simple coal workers' pneumoconiosis, a summary of chest x-ray interpretations would be included only if the x-ray was interpreted as revealing large opacities indicative of complicated pneumoconiosis. None of the chest x-ray evidence in the record revealed large opacities.

Pulmonary Function Studies

The record contains the following pulmonary function study evidence:

Ex. No.	Date	Age	Height	FEV1	MVV	FVC	FEV1/FVC%	Qualify?
DX 42	2-4-87	55	67.5"	2.60	79	3.16		No.
DX 42	11-17-87	55	67"	2.72	81	3.68		No.
				*2.84	*73	*3.82		No.
DX 42	11-9-88	56	67"	2.74	93	3.71		No.
DX 14	2-4-00	68	66.75"	1.76	55	2.93		No.
DX 36	3-22-01	69	65.5"	1.74	--	2.79	62%	No.
				*1.75	*--	*2.73	*64%	No.
DX 58	6-7-01	69	65.5"	0.97	42	1.55	63%	Yes.
				*1.77	52	2.79	63%	No.

* = Post-Bronchodilator

Arterial Blood Gas Studies

The record contains the following arterial blood gas study evidence:

Ex. No.	Date	pO2	PCO2	Qualify?
DX 42	2-11-87	80.2	32.4	No.
		*79	*33.8	No.
DX 42	11-17-87	78	33	No.
DX 16	12-4-00	69	45	No.
		*65	*47	No.
DX 36	3-22-01	72	30	No.

* = Post-Exercise

CT Scans

Dr. Fino's interpretations of the CT scans taken December 15, 2000 and February 19, 2001 were normal. (DX 36). Dr. Wiot found no evidence of coal workers' pneumoconiosis on the CT scans of December 15, 2000 and February 19, 2001. (DX 41). Dr. Shipley found no evidence of CWP but detected tiny scattered nodules that he opined likely reflect healed granulomatous disease on the December 15, 2000 and February 19, 2001 CT scans. (DX 41). Dr. Castle's interpretations of the CT scans taken December 15, 2000 and February 19, 2001, revealed no evidence of CWP. Dr. Hussein's impression of the February 19, 2001 CT scan was as follows:

> Evidence for previous granulomatous disease with calcified mediastinal and hilar nodes; calcified splenic granulomata; previously described nodule in the right upper lobe is reidentified, measuring approximately 5.0 x 3.0 mm; this may have enlarged in size; this may also relate to technical differences as well as intrinsic small size of the lesion; two additional nodular lesions are seen in the right mid to lower lung field, each measuring approximately 5.0 mm; these were not seen previously; this may relate to intrinsic small size of the lesions; multiple additional subpleural nodules bilaterally which are subcentimeter in size; these may relate to previous granulomatous disease, although early metastatic disease cannot be entirely excluded. (DX 103).

Dr. Jonathan Abrahams disagreed with Dr. Hussein's interpretation. His interpretation of the February 19, 2001 CT scan was that the findings are consistent with progression of the miner's granulomatous process or even suggest the possibility of early metastatic disease. He noted that the "subpleural nodules" are probably related to the vascular enhancement pattern

seen against a background of obstructive lung disease. (DX 103). Dr. Harron detected interstitial fibrosis consistent with pneumoconiosis, calcified plaque consistent with asbestos, and cardiomegaly on the February 19, 2001 CT scan. (DX 57).

Medical Reports

Hospitalization Records and Treatment Notes

The record contains records from Windber Hospital. The miner was admitted on February 10, 1982. (DX 42). The admission impression was: Acute transmural inferior infarction complicated by ventricular fibrillation. All of the risk factors for coronary disease, past history of peptic ulcer, questionable chronic GI bleeding, history of heavy alcohol intake. The discharge diagnosis was primary musculoskeletal chest pain with no evidence of reinfarction and the secondary diagnosis was coronary artery disease s/p acute inferior myocardial infarction.

The record contains progress notes from the miner's treating physician, Dr. Jerry Gray, from the period of March 17, 1982 until February 1999. (DX 33). The miner was basically followed for his post-heart attack and the notes track occasional pain determined to be epigastric. The notes reveal that the miner was virtually asymptomatic, with exception of weight gain noted in January 1997, February 1998, and back pain in 1999. Dr. Gray noted his cholesterol had gone up and commented that he was concerned about gathering risks for recurrent MI.

The record contains the progress reports and treatment notes of Dr. Jerry Gray ranging from February to November 2001. (EX 103). The legible sections of the records indicate that the miner was seen for regular visits and follow-up for problems of isolated pulmonary nodule, fatigue, coronary heart disease, and shortness of breath. In April of 2001, Dr. Gray noted that the miner was seen in follow up for isolated pulmonary nodule, fatigue, coronary heart disease, and shortness of breath. A repeat CAT scan showed no progressions and the miner was completely asymptomatic and denied chest pain, shortness of breath, or any new symptoms. On November 14, 2001, the miner reported being reasonably active and virtually asymptomatic, aside from shortness of breath on exertion. Dr. Gray noted that the miner was still too heavy and has done better than he had ever hoped.

The miner was hospitalized on February 2, 2002 at Windber Medical Center. (EX 104). The miner was seen in the ER where he was brought in for cardiac arrest. The treatment report records of Dr. Ahmad indicate that the miner was brought in by ambulance in caradiac arrest. He noted that the miner's wife and paramedics stated that the miner was fine and suddenly held his chest, passed out, and became unresponsive. The family started CPR, the paramedics arrived within five to ten minutes and found the miner to be apneic, cyanotic, and in V-fib. The miner was defibrillated 5-6 times, intubated, and went into asystole. The miner was given Epinephrine and Atopine and an external pacemaker was tried. By the time the miner was brought to the ER he had been down for 40 minutes. On arrival to the ER, he was completely asystolic, pulseless, apneic, and cyanotic, pupils were fixed and dilated. Dr. Ahmad's diagnosis was cardiopulmonary arrest most likely acute cardiac event, dysrrhythmia, V-tach,V-fib.

Physician Opinion Reports

Dr. Robert F. Klemens

Dr. Klemens examined the miner, performed objective tests, and drafted a report on February 4, 1987. (DX 42). Dr. Klemens recorded nineteen years of coal mine employment and noted the miner worked as a continuous miner operator, belted, and helped load coal in cars. He recorded a family history that was positive for stroke in his mother, who died at age forty nine. He noted that the miner reported having pneumonia at age nineteen, attacks of wheezing, and a heart attack in 1982. Dr. Klemens recorded a cigarette smoking history of one half pack per day for 25 years, ending in 1977. He noted that the miner reported cough, sputum, and wheezing for ten to eleven years, and dyspnea with exertion about fifteen years. Physical exam comments were that lungs were clear to percussion and auscultation and no rales or wheezes were heard. Dr. Klemens' diagnosis was coal workers' pneumoconiosis 1/1, p/s (illegible) that is a result of twenty years of coal mining.

Dr. Joseph T. Sobieski

Dr. Sobieski, who is board certified in internal medicine, examined the miner on November 17, 1987 and submitted a report dated November 30, 1987. (DX 42-28). Dr. Sobieski recorded a coal mine employment history of approximately nineteen years, noted the miner's various jobs, and recorded that he was required to lift bags of rock dust weighing fifty pounds. Dr. Sobieski recorded the miner's complaints as shortness of breath while walking up ten stairs and that the miner can walk several blocks without shortness of breath. He recorded that the miner reported getting short of breath about two years before leaving the mines. He noted that the miner's hobbies included walking for exercise and fishing. Dr. Sobieski recorded a smoking history that ended ten years prior and that the miner smoked one half pack of cigarettes per day for twenty years.

Dr. Sobieski noted that the miner had a heart attack in 1982 and is treated by Dr. Jerry Gray. Physical examination revealed normal respiratory pattern without intercostal reactions, pursed lip breathing, or use of the accessory musculature. He recorded that the lungs were clear to auscultation and percussion and there was no cyanosis or clubbing of the fingernails. The miner's heart was normal with a left sided S4 gallop. He noted no pitting edema. The miner's chest x-ray showed no pneumoconiosis, his ventilatory study and arterial blood gas study were normal. The miner's EKG revealed an old inferior wall myocardial infarction.

Dr. Sobieski opined that because of the normal physical exam and diagnostic tests, there was no evidence of respiratory impairment. He opined that the miner's subjective symptoms of occasional wheezing, productive cough would be due to smoker's bronchitis consistent with his long smoking history. Dr. Sobieski opined that the miner's coronary artery disease would cause the dyspnea symptoms and he would be able to continue his previous coal mining job.
Dr. Sobieski stated that it is possible he had a disability because of coronary artery disease but he needs further testing to confirm it.

Dr. Gregory J. Fino

Dr. Fino first reviewed the miner's records and submitted consultative report on October 12, 1988 in connection with his previous claim. (DX 42). Dr. Fino is board certified in internal medicine, pulmonary disease, and is a B-reader of x-rays. (EX 4).[5] Dr. Fino opined that there is insufficient evidence to justify a diagnosis of simple coal workers' pneumoconiosis and there is no respiratory impairment. Dr. Fino opined that from a respiratory standpoint, the miner is neither partially nor totally disabled from returning to his last job in the mines or a job requiring similar effort. He stated that looking at the miner as a whole, he might be disabled due to cardiac disease but the cardiac disease was neither caused nor aggravated by coal dust inhalation. Dr. Fino opined that the miner's disability was neither caused in whole nor in part by coal dust inhalation or coal workers' pneumoconiosis.

Dr. Fino examined the miner on March 22, 2001. This report appears in the record at DX 36. Dr. Fino opined that there is insufficient objective medical evidence to justify a diagnosis of simple CWP and that the miner did not suffer from an occupationally acquired pulmonary condition. Dr. Fino stated that there is a disabling respiratory impairment present due to smoking and that even if he were to assume the presence of radiographic pneumoconiosis, the miner would still be as disabled as he finds him now. Dr. Fino opined that based on all of the information he reviewed, it is his opinion that the miner's respiratory abnormality was not caused, in whole or in part, by the inhalation of coal mine dust.

Dr. Fino's deposition was taken on January 16, 2002 and the deposition transcript appears in the record at DX 61. He testified that he assesses impairment from a pulmonary standpoint by utilizing the objective tests of lung function and that patient complaints do not factor in assessing impairment because it has been clearly shown in the medical literature that there is no correlation between a patients symptoms and impairment. (DX 61 at p. 8). He explained that when a patient complains of dyspnea, the physician must determine the cause. (DX 61 at p. 9). Dr. Fino testified that a significant side effect of Inderal is obstructive lung disease, worsening of pre-existing obstructive lung disease and blunting or preventing a bronchodilator response. (DX 61 at p. 15). Dr. Fino testified that the exercise tests in the records indicate a deconditioning type of abnormality and that the miner exercised to 10.1 METs, which is very heavy labor. (DX 61 at p. 17).

Dr. Fino testified that the miner's spirometry showed moderate obstruction and that the miner had an amazing bronchodilator response. (DX 61 at pp. 18-19). He opined that from a pulmonary standpoint, the miner could not return to his last coal mine job, which involved heavy labor. (DX 61 at p. 20). He opined that even though the miner's coal mine dust exposure was sufficient to explain his disability, he thinks the time line of events do not support it as the cause. (DX 61 at p. 21). He testified that he cannot pinpoint the etiology and before Dr. Schaaf's report, would not have considered asthma because no other studies showed the bronchodilator response. (DX 61 at p. 22). Dr. Fino testified that if he were the miner's treating physician, he would wean him off of Inderal and if coal dust induced disease were the cause of the miner's

[5] Dr. Fino became a B-reader in February 1989, after this examination of the miner. His other credentials were in effect at the time of this examination. (EX 4).

disability, one would not see an improvement. (DX 61 at p. 23). Dr. Fino testified that he would disagree with Drs. Castle and Rosenberg that the miner was not disabled. (DX 61 at p. 26).

Dr. Fino reviewed additional medical information and submitted a report dated January 5, 2004, which appears in the record at EX 3. In connection with this report, Dr. Fino reviewed Windber medical records, the records of the miner's terminal emergency room visit, the autopsy report and death certificate, and the pathology reviews by Drs. Bush, Oesterling, and Perper. Dr. Fino explained that the recent information documents the presence of simple coal workers' pneumoconiosis; therefore, he would change his previous diagnoses and opines that the miner had an occupational lung disease.

Dr. Fino stated that even assuming all of the miner's obstruction and disability were due to coal mine dust, it still does not indicate that coal mine dust played a role in his death. Dr. Fino discussed the terminal emergency room records and explained that they reveal a classic clinical presentation for an acute cardiac event that was subsequently shown pathologically based on evidence of old and new myocardial infarctions and pulmonary edema within the lung tissue. Dr. Fino disagreed with Dr. Perper's report and stated that there is no evidence that the miner had any form of hypoxemia at or about the time he died, and that there is ample medical literature showing that hypoxemia in coal workers' pneumoconiosis does not cause coal miners to die more frequently than non-miners as a result of heart disease.

Dr. Fino emphasized that there was no evidence of hypoxemia occurring at the time the miner died and explained that the most recent arterial blood gas study in March 2001 was normal. He explained that even though mild resting hypoxemia was present in December 2000, it was chronic hypoxemia that was reversible and patients with chronic hypoxemia do not die more frequently as a result of that chronic hypoxemia, and noted that Dr. Perper did not provide objective evidence to support this hypothesis.

Dr. Fino stated that it is pure speculation on Dr. Perper's part to state that the miner's direct pulmonary insufficiency or hypoxemia was a participating factor in his death. He explained that the pathology and clinical history show that the miner's death was a case of primary cardiac disease and that the suffered a heart attack due to underlying heart disease. Dr. Fino noted that the miner had a witnessed cardiac arrest outside of the hospital and opined that the miner's death was unrelated to the inhalation of coal mine dust, even if he were to assume that all of the lung disease was due to coal mine dust. Dr. Fino opined that regardless of the amount of lung disease present or the cause of that lung disease, the miner would have died in the same manner, fashion, and at the same time had he never stepped foot in the coal mines. He opined that coal mine dust inhalation did not cause, contribute to, or hasten the miner's death.

Dr. Vijay Malhotra

Dr. Malhotra examined the miner on November 9, 1988 and submitted a report dated December 9, 1988. (DX 42). Dr. Malhotra noted that the miner worked nineteen years as an underground coal miner and described his work scooping up coal, hauling supplies and fifty pound bags of rock dust in a very dusty environment. Dr. Malhotra recorded that the miner complained of shortness of breath times ten years, which is getting progressively worse. He

recorded a smoking history of one pack per day for twenty six years, ending ten years prior. Physical examination revealed no ankle edema, cyanosis, or clubbing, and the lungs were clear on auscultation. He noted that the heart had a regular rhythm and both heart sounds were heard normally. The echocardiogram performed in his office suggested a remote inferior wall myocardial infarction. Ventilatory studies revealed mild reduction in MVV, and the FEV1 and FVC were 87% and 93% of normal respectively. Dr. Malhotra noted that although the miner had advanced respiratory symptoms, he had no physical, chest x-ray, or pulmonary ventilation studies suggestive of advanced pneumoconiosis or any pulmonary disability. Dr. Malhotra recommended that the miner be re-evaluated in a few years and emphasized that pneumoconiosis is a progressive disease.

Dr. Glicerio Ignacio

Dr. Ignacio examined the miner on December 4, 2000. (DX 15). He referenced the miner's occupational history form for his work history. Dr. Ignacio recorded a smoking history of one-half to one pack of cigarettes per day from 1952 until the 1970s. He recorded a family history that was positive for stroke in the miner's mother. He recorded the miner's history as positive for pneumonia, wheezing, heat disease, allergies, and high blood pressure and noted that the miner had a heart attack in 1982. Dr. Ignacio noted that the miner reported daily sputum production, daily wheezing, dyspnea since 1979, and cough. The miner's physical exam was essentially normal. The miner's ventilatory study revealed mild to moderate restrictive lung disease and the arterial blood gas study showed mild hypoxemia at rest and exercise. Dr. Ignacio's cardiopulmonary diagnosis was: nodular density at mid-right lung, peripheral portion. The etiology of the cardiopulmonary diagnosis were: 1) History of coronary artery disease; 2) History of hypertension; 3) Cardiomegaly. Dr. Ignacio listed the miner's impairments as: 1) Hyperlipidemia; 2) No pneumoconiosis; 3) No impairment. He noted that the miner should be referred to another physician for evaluation.

Dr.John T. Schaaf

Dr. Schaaf, examined the miner on June 7, 2001 and his report appears in the record at DX 58. Dr. Schaaf performed objective tests that included an EKG, ventilatory study, arterial blood gas study and chest x-ray. Dr. Schaaf recorded that the miner complained of shortness of breath time twenty years and worsening. He recorded at cigarette smoking history of one half pack per day for twenty five years and calculated it to be a ten to twenty pack-year history. He recorded an approximate twenty year underground coal mine employment history and recorded that the miner worked as a roof bolter and unitrack operator. Dr. Schaaf noted that the miner stopped working in 1980 when the mine shut down.

Dr. Schaaf noted on physical examination that breath sounds were normal but remarked that it is difficult to get the miner to do a consistent breathing pattern and that the miner has almost disconnected breathing on command but while at rest and when he is sitting there, he does not seem to have any breathing distress at all. Dr. Schaaf recorded that the miner complained of dyspnea. Dr. Schaaf stated that he suspects the miner does indeed have early coal workers' pneumoconiosis and further manifestations of this included the drop in PO2 during exercise and the other chest x-rays, which were read positive for pneumoconiosis. Dr. Schaaf

stated that his borderline x-ray does not officially support a finding of pneumoconiosis but he believes that the miner has pneumoconiosis. Dr. Schaaf opined that the miner is disabled from his coal mine employment because of his pulmonary problem, which he opined is due to pneumoconiosis.

Dr. James R. Castle

Dr. Castle reviewed the miner's medical records and provided a report dated September 19, 2001, which appears in the record at DX 50 and 58. Dr. Castle is board-certified in internal medicine and pulmonary diseases and is a B reader. Dr. Castle opined, with a reasonable degree of medical certainty that the miner does not suffer from coal workers' pneumoconiosis based upon the chest x-ray reports, physical findings, physiologic findings, or arterial blood gas findings. He opined that the miner is most likely not permanently and totally disabled as a result of any pulmonary process. Dr. Castle noted that although the miner had some variability in his efforts, when valid physiologic studies were obtained, he was above the accepted level of physiologic function indicating disability in coal miners by the Department of Labor. Dr. Castle opined that the miner retained the respiratory capacity to perform his usual coal mining duties.

Dr. Castle stated that it is entirely possible that the miner is disabled as a result of his coronary artery disease, age, and deconditioning, but that these conditions are not related to the inhalation of coal mine dust. Dr. Castle explained that even if he were to conclude that the miner has radiographic evidence of simple coal workers' pneumoconiosis, his opinion concerning his lack of impairment and disability due to that process would remain unchanged. He stated that his opinion is not predicated on the miner having a normal or negative x-ray but is contingent on his not having the physiologic abnormalities indicating disability due to that process.

Dr. Castle submitted another consulting opinion dated November 28, 2001, which appears in the record at DX 58. He stated that it continues to be his opinion, within a reasonable degree of medical certainty, that the miner does not suffer from coal workers' pneumoconiosis. Dr. Castle further opined that the miner is most likely not permanently and totally disabled as a result of any pulmonary process. He stated that while the miner did have variability in his pulmonary function studies, when best efforts are reviewed, they are above federal disability standards. Dr. Castle stated that it continues to be his opinion that the miner may be disabled as a result of other medical problems including his age, but none of the conditions are related to the accumulation of coal mine dust.

Dr. Castle reviewed additional medical records, including his own prior reports, the reports of the other consulting physicians, the death certificate, and the autopsy protocol, and submitted another report dated December 31, 2003. This report appears in the record at EX 1. Dr. Castle opined, with a reasonable degree of medical certainty, that the miner did have pathologic evidence of simple coal workers' pneumoconiosis and evidence of centrilobular emphysema. Dr. Castle stated that it is apparent from the medical records that the miner had generally a normal degree of oxygenation and on one occasion had a PO2 as low as 65mmHg. He explained that this does not represent a significant degree of hypoxemia and this degree of PO2 would not be associated with the development of cardiac arrhythmias. Dr. Castle stated that

events surrounding the miner's sudden death do not indicate the development of an exacerbation of COPD since he was "feeling well" and suddenly grabbed his chest and collapsed. Dr. Castle explained that this indicates that the miner's death was a sudden cardiac event and there is no evidence that hypoxemia played a role in precipitating an arrhythmia.

Dr. Castle stated that even though the miner had pathologic evidence of simple coal workers' pneumoconiosis and underlying pulmonary emphysema, it is his opinion with a reasonable degree of medical certainty that the miner's death was due to cardiac disease and was unrelated to any lung disease including coal workers' pneumoconiosis. Dr. Castle stated that there is no evidence in the records to indicate that the miner developed any hypoxemia at the time of his death, and the records also reflect that his oxygenation was generally normal and that he never developed or demonstrated a significant degree of hypoxemia during life. Dr. Castle opined with a reasonable degree of medical certainty that the miner died as and when he would have regardless of the findings of simple coal workers' pneumoconiosis. He explained that he arrived at this opinion because the cardiac death was unrelated to any lung disease and was a sudden event occurring in an individual with very severe coronary artery disease, which are events that are very common in today's society.

Dr. Castle was deposed on January 24, 2005. (EX 6). He testified that based upon the studies he reviewed and the numbers produced, the miner during his lifetime had the respiratory capacity to return to his last usual coal mine job and although he had a mild to moderate degree of obstruction, he was above federal disability levels in terms of his physiologic function. (EX 6 at p. 16). Dr. Castle testified that in his medical opinion, the miner died as a result of ventricular fibrillation occurring in the setting of acute myocardial infarction that after attempted resuscitation resulted in asystole, and died as a result of an acute myocardial infarction resulting in a fatal cardiac arrhythmia. (EX 6 at p. 17). Dr. Castle testified that the miner did not have significant or consistent hypoxemia and the events surrounding his death were the direct result of his acute myocardial infarction and ventricular fibrillation and he does not believe that hypoxemia played any role in that. *Id.*

Dr. Castle testified that when coal workers' pneumoconiosis results in death, he would expect to see significant impairment, chronic persistent hypoxemia, which was not the case, and many individuals require oxygen, which the miner did not. (EX 6 at p. 18). Dr. Castle testified that the miner had atherosclerotic cardiovascular disease and suddenly, without any premonitory symptoms, had a heart attack associated with ventricular fibrillation, and there was no evidence the miner had any progressive hypoxemia. (EX 6 at p. 21).

Dr. Castle testified that the combination of a disrupted quantity and a poor quality of blood getting to the heart can cause a sudden death if those events were taking place. (EX 6 at p. 23). Dr. Castle testified that he believed the miner could have performed the heavy manual labor of his previous employment from a pulmonary standpoint because his pulmonary function studies and arterial blood gas studies did not meet federal disability guidelines. (EX 6 at p. 29). Dr. Castle testified that the miner had some obstructive disease and on one occasion a minimal degree of hypoxemia that was transient in nature and he could still do his work in the mines. (EX 6 at p. 32.

Dr. David M. Rosenberg

Dr. Rosenberg, who is board certified in internal medicine, pulmonary disease, occupational medicine, and is a B-reader, provided a consulting opinion dated December 27, 2001. His report appears in the record at DX 58. Dr. Rosenberg opined, with a reasonable degree of medical certainty, that the miner does not have coal workers' pneumoconiosis. Dr. Rosenberg stated that while the miner has a degree of airflow obstruction, it relates to his past smoking history and not the past inhalation of coal dust exposure. Dr. Rosenberg opined that the miner could perform his previously defined coal mining job or other similarly arduous types of employment.

Dr. Rosenberg provided a subsequent opinion dated January 5, 2004, which appears in the record at EX 2. Dr. Rosenberg reviewed the autopsy reports, Windber Medical Center records, and the reports of Drs. Bush, Oesterling, and Perper. Dr. Rosenberg opined that based on his review of the records, the miner had simple coal workers' pneumoconiosis. Dr. Rosenberg explained that chest x-rays can appear negative even when pneumoconiosis is observed pathologically because roentgenographic abnormalities may be potentially observed only when micronodularity develops. He further explained that CAT scans are more sensitive than x-rays and the miner's CAT scan did not demonstrate pneumoconiosis, which confirms that the miner's pneumoconiosis was mild. Dr. Rosenberg stated that if Dr. Perper's estimation of 40% involvement of nodular pneumoconiosis was valid, the miner's CAT scans would have demonstrated such findings.

Dr. Rosenberg opined that from a functional perspective, the miner's mild pneumoconiosis would not have been associated with any significant respiratory impairment, the miner's mild airflow obstruction became evident many years after he left the mines, and this airflow obstruction was not associated with disabling PO2 values. Dr. Rosenberg explained that centrilobular emphysema, which Dr. Perper attributed to coal mine dust exposure, is classically associated with cigarette smoking, and emphysema developing in relation to coal dust exposure begins in and around the coal macule. He stated that the miner's centrilobular emphysema was not associated with coal pigment deposition. Dr. Rosenberg pointed out that Dr. Perper's references supporting a relationship between centrilobular emphysema and coal mine dust exposure have, for the most part, not been controlled for cigarette smoking.

Dr. Rosenberg stated that the miner died of a cardiac arrhythmia consequent to his underlying coronary artery disease and any mild, simple pneumoconiosis would not have cause or hastened the miner's death. He opined that the miner's mild, simple pneumoconiosis would not have resulted in any clinically significant physiologic impairment or hypoxia and one should appreciate that the miner's arrhythmia came on suddenly. Dr. Rosenberg stated that the records clearly indicate the miner had an evolving myocardial infarction which started days prior to his death, and was similar to what happened in 1982, when the miner had another episode of ventricular fibrillation in relation to a heart attack.

Dr. Rosenberg opined, within a reasonable degree of medical certainty, that the miner had simple pneumoconiosis, which was not causing any significant respiratory impairment, and any impairment present was related to his long smoking history with the development of

centrilobular emphysema, and an asthmatic component. He opined that the miner's death was related to his underlying coronary artery disease, which was not caused or hastened by the past inhalation of coal mine dust or the presence of pneumoconiosis. Dr. Rosenberg opined that the miner developed a fatal arrhythmia related to a myocardial infarction and this same type of arrhythmia previously developed in 1982, also in relationship to a myocardial infarction.

Death Certificate

The miner's death certificate was filed on February 5, 2002 and was completed by Jerry L. Gray, M.D. (DX 101). The immediate cause of death was listed as: a) Acute myocardial infarction; and b) Longstanding coronary artery disease. There were no other significant conditions listed.

Autopsy Prosector's Report—Dr.Waheeb Rikzalla

An autopsy was performed on February 3, 2002. The autopsy prosector was Dr. Rizkalla at Conemaugh Memorial Hospital. The autopsy report appears in the record at DX 67, 72, and 102. The final anatomic diagnoses were listed as: severe atherosclerotic coronary artery disease; old myocardial infarction; cor pulmonale (right ventricle 0.8 cm); moderate pulmonary emphysema; and simple micronodular coal workers' pneumoconiosis. The clinical pathological summary stated, "This 70 year old, white male died from severe atherosclerotic coronary artery disease. Additional findings are simple micronodular coal workers' pneumoconiosis and pulmonary emphysema.

The heart weight 490 grams and the coronary arteries showed severe atherosclerotic narrowing ranging up to 90% of the vessel diameter in the right coronary artery and left descending coronary artery. The myocoardium was firm and red-brown, and showed a tan-white area in interventricular system measuring 1.5 x 1.0 cm. The left ventricle measures 1.7 cm in thickness 2.0 cm below the aortic valve and the right ventricle measures 0.8 cm in thickness, 2.0 cm below the pulmonary valve. The cardiac chambers on the right side were dilated and the valve leaflets were soft and pliable without vegetations. The aorta and its main branches showed severe atheromatous changes without ulcerations. Microscopic visualization revealed dense fibrous scarring involving the left ventricle consistent with an old myocardial infarction. There is calcific obstructive atherosclerotic coronary artery disease involving the left anterior descending and right coronary arteries with significant narrowing up to 90 percent. Intimal thickening of the aorta is present with cholesterol clefts and dystrophic calcification.

The lungs revealed no thromboemboli. On palpation, the hilar lymph nodes were enlarged. The pleural surfaces of the lungs displayed multiple black macules covering approximately 85 percent of the lung surfaces. On palpation, the lungs displayed multiple firm nodules, the largest measuring 0.5 cm in diameter covering approximately three percent of the lung surfaces. When sectioned, the pulmonary parenchyma displayed black macules measuring up to 0.4 cm in diameter involving approximately 75 percent of the lung parenchyma. Microscopic examination of the lungs revealed enlarged air spaces with distal spurring in a centrilobular pattern. Collections of anthracotic pigment laden macrophages were present and macules are seen in a distributive pattern in the bronchial, interstitial, perivascular, and

subpleural areas. The macules displayed associated focal emphysema and reticular fibrosis and range in size up to 0.4 cm and involve 75 percent of the parenchyma. Parenchymal nodules composed of dense fibrocollagenous stroma with scattered anthrasilicotic pigment laden macrophages were seen in similar distribution as the macules. The nodules ranged in size up to 0.5 cm and involve 5 percent of the parenchyma. Pulmonary arteriolar hypertrophy is prominently present and focal collection of noncaseating granuloma were seen. Special stains (PAS, AFB, B& B) were negative.

Dr. Rizkalla Deposition

Dr. Rikzalla was deposed on February 5, 2003 and his deposition appears in the record at EX 5. Dr. Rizkalla testified that he is chairman of the Department of Pathology and Laboratory Medicine at Conemaugh Memorial Medical Center and that he performs approximately fifty autopsies a year, with thirty to thirty five of those pertaining to the presence or absence of coal workers' pneumoconiosis. (EX 5 at pp.7, 11-12). Dr. Rizkalla testified that the miner's autopsy showed changes consistent with simple coal workers' pneumoconiosis, and revealed both macular and micronodular forms of the disease. (EX 5 at p. 13). He explained that both forms were classified as simple coal workers' pneumoconiosis and none of the nodules exceeded 0.7 centimeters. *Id.* Dr. Rizkalla explained that the macular form of simple pneumoconiosis can result in some pulmonary impairment with other associated lesions of the macules such as emphysema, and impairment is more often associated with the micronodular form of the disease. EX 5 at p. 15.

Dr. Rizkalla testified that the immediate cause of death was the miner's severe atherosclerotic coronary artery disease and that his moderately severe centrilobular emphysema associated with the coal workers' pneumoconiosis was a substantial contributing factor in the mechanism of death. (EX 5 at p.21). He explained that he reached that conclusion because the miner had severe upper sclerotic coronary heart disease, which had nothing to do with his occupation, but the right ventricle of the heart was dilated and thickened twice the normal level, so by definition, he had cor pulmonale. (EX 5 at pp. 21-22). Dr. Rizakalla explained that the miner had centrilobular emphysema, due in part to smoking, and exposure to coal dust and simple CWP, which is also known to induce centrilobular disease. (EX 5 at p. 22). He stated that the severe atherosclerotic heart disease would narrow the primary vessels and increase the heart mass, reducing the amount of blood, and the quantity of blood coming through the arteries was not optimal. (EX 5 at pp. 22-23). Dr. Rizkalla explained that a normal person with a normal coronary system had an oxygen saturation of 70, like the miner, might be okay, but with the miner's narrow blood vessels, the amount of blood is reduced, the minimum reduction of oxygen saturation coming to his heart can precipitate the patient to develop a cardiac arrhythmia. (EX 5 at p. 23).

Dr. Rizkalla testified that the miner weighed between 250 to 300 pounds and was overweight. (EX 5 at p. 26). He testified that autopsy revealed evidence of a previous infarction on the heart in the left ventricle. (EX 5 at p. 28). Dr. Rizkalla testified that cor pulmonale was present and he made that diagnosis in relation to the right ventricular thickening with the dilatation. (EX 5 at p. 32). Dr. Rizkalla testified that the right coronary artery was 90% occluded which is a significant finding and explained that once it's blocked, it induces death to that area

where it supplies the heart. (EX 5 at p. 34). Dr. Rizkalla testified that he did not see any evidence of a recent heart problem and disagreed with Dr. Oesterling's observation of a recent myocardial infarction because he believes Dr. Oesterling was describing the presence of collagen, which indicates an old process. (EX 5 at p. 46-47). Dr. Rizkalla agreed with Dr. Oesterling that the miner had significant ischemic myocardiopathy, but disagreed that it resulted in severe passive congestion with pulmonary edema because he did see that the alveolar spaces of the lungs filled with macrophages, as expected. (EX 5 at pp. 48-49).

Dr. Rizkalla testified that he cannot determine whether cigarette smoking or coal mine dust exposure led to the miner's centrilobular emphysema. (EX 5 at p. 50). He testified that patients without any lung disease die as a result of cardiac disease when they have coronary arteries as diseased as the miner's were. (EX 5 at p. 53). Dr. Rizkalla testified that he expects to see cor pulmonale in cases of moderately severe coal workers' pneumoconiosis and that the miner had moderate simple coal workers' pneumoconiosis. (EX 5 at p. 55). He testified that the thickening of the pulmonary arterial branches indicate that the right ventricle was pumping the blood against pressure, which is another term for pulmonary hypertension, which one can also see in coal workers' pneumoconiosis. (EX 5 at p. 63). Dr. Rizkalla testified that he agrees with Dr. Oesterling that the right ventricle can contain some fatty cells but stated that the fat infiltration will not increase the thickness up to eight millimeters, as in the miner's pathology, which is twice normal and that the presence of fat in the right ventricle does not mean that the patient does not have cor pulmonale. (EX 5 at pp. 64-65). Dr. Rizkalla testified that Dr. Perper's finding of thickening of the intrapulmonary blood vessels will be reflected to the right ventricle, inducing the cor pulmonale. (EX 5 at p. 67-68). Dr. Rizkalla testified that if the miner's right ventricle was not thickened, the right ventricular wall would have been greater. (EX 5 at p. 69).

Dr. Rizkalla testified that the miner had scarring of the left ventricle. (EX 5 at p. 75). Dr. Rizkalla explained that the difference between his measurement of the largest nodule (5mm) and Dr. Perper's measurement (8mm) could be a result of the shape of the nodule. (EX 5 at 76). Dr. Rizkalla testified that he would not have opined that pneumoconiosis or emphysema contributed to the miner's death in the absence of cor pulmonale and it is unimaginable that the miner would have that amount of coal dust in his lungs and macules without inducing the cor pulmonale. (EX 5 at p. 84).

Dr. Stephen T. Bush

Dr. Bush, who is board certified in anatomic and clinical pathology, reviewed the miner's medical records and provided a consulting opinion dated September 27, 2002. His report appears in the record at DX 72 and 105. Dr. Bush opined that the miner had a mild degree of simple coal workers' pneumoconiosis that did not cause respiratory impairment or disability during the miner's lifetime. He noted that the lung slides have 1-2 coal worker micronodules ranging from 0.1-0.3 cm, which consist of black dust pigment consistent with coal dust free in the tissue and in macrophages associated with a fibrous reaction. Dr. Bush noted that less than three percent of the lung parenchyma is disrupted by the coal worker lesions, which contrasts with the autopsy report describing seventy five percent involvement of the parenchyma. Dr. Bush also noted larger nodules representing granulomas resulting from a fungal infection in

the distant past and correspond with those seen by the radiologists. He noted that they measure up to 0.8 cm.

Dr. Bush stated that the slides showed a variable amount of centrilobular emphysema that is not associated with coal worker lesions or coal dust pigment but is typical of that found in cigarette smokers.

He explained that the pneumoconiosis was too limited in degree and extent to have caused any respiratory impairment or disability. Dr. Bush stated, with reasonable certainty, that the cause of death is cardiac disease. He explained that medical information documents severe coronary artery disease with past myocardial infarction. Dr. Bush also noted that the past history of smoking, which is causally related to coronary artery disease, and the miner's significant obesity that was noted at autopsy, make a cardiac cause of death highly probable. Dr. Bush further opined that death was not caused by, contributed to, or hastened by any chronic dust disease arising from coal mine employment and the degree of chronic dust disease related to coal mine employment is very limited and could not have been a factor in the events leading to death.

Dr. Bush testified in a deposition on February 7, 2005. (EX 8). He testified there was nothing in the miner's case indicating progressive massive fibrosis or complicated pneumoconiosis. (EX 8 at pp. 32-33) Dr. Bush testified that the extent of the lung substance damage by coal workers' disease totaled no more than three percent of the lung tissue. (EX 8 at p. 45). He explained that he took Dr. Rizkalla's estimate of 75% of the lung to indicate that he was looking at a discolored lung. *Id.* Dr. Bush testified that he saw centrilobular emphysema that was quite variable and not related to the coal dust pigment or coal dust lesions specifically. (EX 8 at p. 48). Dr. Bush testified that the miner's heart was enlarged, his left ventricular muscle wall was thicker than normal, and the right ventricular wall was 0.8 cm and the normal would be about 0.5 cm. (EX 8 at pp. 50-51). Dr. Bush stated that Dr. Rizkalla was describing biventricular hypertrophy, enlargement of the heart involving both the left and right ventricles that is typically found in individuals with some element of chronic hypertensive disease. (EX 8 at p. 51).

Dr. Bush testified that he saw no evidence of pulmonary hypertension and he did not find any evidence of cor pulmonale. (EX 8 at p. 54). He stated that there was thickening of the right ventricle, but that is not the same thing as saying there is cor pulmonale. Id. He testified that to make the diagnosis of cor pulmonale, one would expect significant disease involving the lungs that could produce obstruction of blood flow to the degree that arterial pressure would rise. (EX 8 at 55-56). Dr. Bush testified that cor pulmonale would typically produce abnormal pulmonary function studies. (EX 8 at p. 57). Dr. Bush testified that he did not ignore the autopsy findings of cor pulmonale but did not discuss them in his report because it was absent. (EX 8 at 57-58). Dr. Bush explained that if there was a progression of pneumoconiosis in the miner, it did not progress very far because he did not have severe disease. (EX 8 at p. 58).

Dr. Bush testified that most centrilobular emphysema studies cited by Dr. Perper ignore the focal emphysema that occurs with coal worker lesions. (EX 8 at pp. 59-60). Dr. Bush testified that there was not much major pathology in the miner and he had emphysema and coal workers' disease of a mild degree and extent. (EX 8 at p. 61). Dr. Bush testified that the miner died of his known cardiovascular disease that produced sudden, unexpected death and he did not

find evidence that his coal mine dust induced disease caused or contributed to a lifetime impairment or hastened his demise. EX 8 at pp. 62-63. Dr. Bush testified that it is by omission that he indicated in his report that the macular disease was not a significant presence. (EX 8 at p. 66).

Dr. Everett F. Oesterling

Dr. Oesterling reviewed the miner's medical records and submitted a report dated November 6, 2002, which appears in the record at DX 73,122. Dr. Oesterling is board certified in anatomical pathology, clinical pathology, and nuclear medicine. Dr. Oesterling reviewed the autopsy slides and stated with a reasonable degree of medical certainty that: 1) there is evidence of a mild macular with micronodular coalworkers' pneumoconiosis; 2) the limited structural change that results from this degree of mild dust deposition is insufficient to have altered pulmonary function, and it would not produce respiratory disability; 3) the changes are insufficient to have in any way contributed to, hastened, or caused the miner's death. Dr. Oesterling noted that the miner had passive congestion of the lungs that is seen in a failing heart as a result of severe arteriosclerosis involving the left descending coronary artery, which is hardening of the arteries.

Dr. Oesterling stated that photo 29 revealed collagen fibers with relatively young areas of fibrosis, probably one to two months old. He noted that other fibers are fragmented and surrounded by clear halos indicating shrinkage and have died approximately four to six days before the miner's death. Dr. Oesterling stated that the photos reveal that the miner was experiencing significant ischemic cardiomyopathy during his terminal days and weeks, the latter having resulted in severe passive congestion with pulmonary edema, which would lead to cardiac arrhythmia, cardiac arrest and death.

Dr. Oesterling noted that photo 36 revealed adipose cells indicating diffuse infiltration of the right ventricular wall with fat, that the miner was obese, which impacted on his heart by producing fatty infiltration of the myocardium. He explained that the right ventricular measurement of 0.8 cm described by the prosector as cor pulmonale is not muscular hypertrophy but is fatty infiltration, unrelated to any degree of pulmonary hypertension attributed to pulmonary disease. Dr. Oesterling noted the miner's mother and two brothers who suffered from myocardial infarctions.

Dr. Oesterling stated that multiple factors associated with the evolution of heart disease are clearly presented within the miner's medical records. He opined that coal workers' pneumoconiosis is not a factor in the evolution of coronary arteriosclerosis and resultant ischemic myocardiopathy, and it did not contribute to, hasten, or cause the miner's death.

Dr. Oesterling was deposed on February 9, 2005. (EX 7). He testified that he is chairman of the department of pathology at Ohio Valley General Hospital. (EX 7 at p. 4). Dr. Oesterling testified that he agreed with Dr. Rizkalla that the miner had both macular and micronodular coal workers' pneumoconiosis but he himself did not see macular involvement of 75% of the lung parenchyma permeated with black pigment. (EX 7 at pp. 18-19). Dr. Oesterling testified that he did not see a causal association with coal dust and the areas of centrilobular emphysema because

there is not black pigment involved in the areas of emphysema. (EX 7 at p. 32). He explained that emphysema in coal miners is typically due to industrial bronchitis occurring during the active inhalation of dust and that the macular lesions on the photographs revealed enlarged airspaces of focal emphysema, which is typically associated with the deposition of coal mine dust. (EX 7 at pp. 32-33). Dr. Oesterling described the presence of atelectasis, caused by the failing left heart ventricle in the photographs. (EX 7 at p. 35).

Dr. Oesterling testified that the miner's heart was abnormally enlarged on both sides and explained that the miner gained 100 pounds over a relatively short period of time and the sudden ontake of weight resulted in fat storage in his right ventricle. (EX 7 at p. 37). Dr. Oesterling described ghost fibers visible in the photographs where very little substance is in the fiber and explained that it has died, the protein is removed, the nucleus is gone, but the membrane persists, resulting in a ghost cell. (EX 7 at p. 40). He explained that from this, a pathologist can conclude that that portion of fiber is dead and has been dead at that stage two weeks or greater. (EX 7 at p. 41). Dr. Oesterling testified that the miner had some precursor events before his death, including increased shortness of breath. *Id.* He explained that the heart was no longer able to function well because of infiltration in the wall. *Id.*

Dr. Oesterling testified that he found evidence of the miner's previous heart attack. (EX 7 at p. 42). He testified that he classified the miner's pneumoconiosis as a relatively mild macular and micronodular coal workers' pneumoconiosis. (EX 7 at p.43). Dr. Oesterling explained that that he does not believe the level of disease in the miner's lungs produced any significant alteration in function and explained that the air spaces he previously mentioned alter lung function, as opposed to macular surrounding vessels or the micronodules. *Id.* Dr. Oesterling opined that the miner died due to progressive ischemic myocardiopathy, causing progressive death in his heart which reached a critical phase and he went into cardiac arrest and death. He stated that it was complicated by the passive congestion in the lungs which resulted from the failing left ventricle. (EX 7 at pp. 43-44). He opined that the miner's work or coal workers' pneumoconiosis did not contribute, cause, or hasten his death, and he believes his demise was due to multiple other factors. (EX 7 at p. 45).

Dr. Oesterling testified that he would estimate that two percent maximum of actual structural change due to dust deposits and approximately ten to fifteen percent to emphysema. (EX 7 at p. 48). Dr. Oesterling testified that hypoxemia was present at the time of the miner's death and explained that most of it was experienced after his initial heart attack and that he also had emphysema. EX 7 at pp. 48-50. Dr. Oesterling testified that the miner had a fatty right ventricle and a slightly enlarged left ventricle, and an enlarged heart. (EX 7 at p. 57). Dr. Oesterling testified that an individual with cor pulmonale usually has a blood gas abnormality but the major change is pressure. (EX 7 at p.59).

Dr. Joshua A. Perper

Dr. Perper, who is board certified in anatomical and forensic pathology and is Chief Medical Examiner of Broward County, Florida, reviewed the miner's records and submitted a report dated November 27, 2002. (DX 75). Dr. Perper opined that the miner had evidence of long standing occupational exposure to coal mine dust, and clinical and radiological findings

consistent with the autopsy findings of severe coal workers' pneumoconiosis with associated centrilobular emphysema. He stated that the miner, with an occupational exposure of more than nineteen years of underground coal mining, developed substantial and significant coal workers' pneumoconiosis as a result of his occupational exposure to coal mine dust. Dr. Perper opined that coal workers' pneumoconiosis with associated centrilobular emphysema was the primary cause of the miner's total and permanent pulmonary disability that prevented him from returning to his regular coal mining work or similar physical work. Dr. Perper also provided citations to medical literature and background information on coal workers' pneumoconiosis and associated centrilobular emphysema.

Dr. Perper reviewed additional records and submitted another report dated April 8, 2003, which appears in the record at DX 82 and 125. Dr. Perper stated that the miner had evidence of significant and substantial coal workers' pneumoconiosis, causally associated with centrilobular emphysema, and that the miner's coal workers' pneumoconiosis was the result of more than twenty years of occupational exposure as a coal miner to coal dust containing silica, a more than sufficient exposure period necessary for developing coal workers' pneumoconiosis. Dr. Perper opined that coal workers' pneumoconiosis and the associated centrilobular emphysema was a substantial contributory cause of the miner's death, both directly and indirectly through pulmonary insufficiency and through hypoxemia, triggering or aggravating an arrhythmia, on the background of marked heart disease. Dr. Perper also provided citations to medical literature and background information on coal workers' pneumoconiosis and associated centrilobular emphysema.

Dr. Perper was deposed on May 9, 2003. (DX 82, 126). He testified that in his opinion, part of the miner's centrilobular emphysema was a result of smoking. (DX 126 at p. 15). He testified that most of the pulmonary function studies showed an obstructive defect, although Dr Schaaf's studies also showed a restrictive defect, which he agrees with. (DX 126 at pp.21-22). Dr. Perper testified that the miner's arterial blood gas studies showed mild hypoxemia and were slightly abnormal. (DX 126 at pp.23). Dr. Perper acknowledged that the miner was asymptomatic in his treating physician exams while reporting dyspnea with Dr. Klemens, and surmised that it could be that the miner was not asked about shortness of breath or that he had lived with it for so long it was not necessary to mention it to his treating physician. (DX 126 at pp. 26-28). Dr. Perper agreed that during this same period, Dr. Malhotra performed objective studies that were virtually normal. (DX 126 at p. 29).

Dr. Perper testified that the miner's weight recorded as 280 pounds on autopsy might be a mistake by the prosector and might have actually been 208 pounds. (DX 126 at p. 37). Dr. Perper testified that once a person stops smoking, any emphysema caused by cigarette smoking stops progressing. (DX 126 at p. 41). Dr. Perper explained that high blood pressure affects the left side of the heart and pulmonary hypertension affects the right side, and in order to have the latter condition there would be severe right heart failure. (DX 126 at p. 44). He testified that the miner did not have pulmonary hypertension during his lifetime. (DX 126 at p. 45). Dr. Perper testified that he did not consider the macular disease significant and would not have expected it to turn up on an x-ray. (DX 126 at pp. 49-50).

Dr. Perper testified that the cause of death was an acute cardiac event associated with both the heart and lung disease and that pneumoconiosis was a substantial contributing cause of death. He stated that the literature shows that people with chronic lung disease and heart disease are at risk of a fatal cardiac arrhythmia triggered through chronic hypoxemia associated with chronic lung disease. (DX 126 at p. 55). Dr. Perper compared a person with heart disease to a person who is limping on ice and if they get a "push" from hypoxia due to chronic lung disease, it is significant. (DX 126 at p. 56). Dr. Perper based his diagnosis of cor pulmonale on the measurements at autopsy and that the intrapulmonary blood vessels show thickening and sclerosis of the wall, which is a finding consistent with pulmonary hypertension and cor pulmonale. (DX 126 at p. 57).

Dr. Perper testified that a normal left ventricle would be about 1.4 or 1.5 cm and a normal right ventricle would be about 0.4 or 0.5 cm and the miner's ventricles were 0.8 cm which he said is definitely indicative of cor pulmonale. (DX 126 at p. 64).

Dr. Christopher J. Begley

Dr. Begley, who is board certified in internal medicine, pulmonary medicine, and critical care medicine, reviewed the miner's medical records and issued a report dated December 20, 2004 that appears in the record at CX 1. Dr. Begley opined, with a reasonable degree of medical certainty, that the miner suffered from coal workers' pneumoconiosis. He based this opinion on the miner's long history of coal dust exposure, multiple abnormal radiographs, and the findings on the autopsy. Dr. Begley noted that the autopsy revealed 75-85% involvement of the pulmonary parenchyma with coal maculae, and 3-5% involvement of his lung parenchyma with fibrocollagenous nodules.

Dr. Begley opined that the miner's coal workers' pneumoconiosis was a substantial contributing factor to, and accelerated the miner's demise. He explained that the miner's coal workers' pneumoconiosis caused significant progressive arterial hypoxemia which worsened his cardiac ischemia. Dr. Begley noted that the miner smoked but quit in the 1970's. Dr. Begley stated that the miner's pneumoconiosis was quite dramatic at the time of autopsy and caused a progressive decline in his respiratory status. Dr. Begley stated that the miner's progressive coal workers' pneumoconiosis is the sole contributing cause to his progressive decline in pulmonary function and progressive arterial hypoxemia. He stated that the increased myocardial oxygen consumption due to increased worsening of breathing, as well as the progressive arterial hypoxemia contributing to cardiac ischemia, and his arterial sclerotic cardiovascular disease led to the miner's ultimate demise.

Dr. Begley was deposed on March 16, 2005. (CX 3). He testified that he is Director of Pulmonary Rehabilitation at Memorial Medical Center, Director of Respiratory Care Services and Pulmonary Rehabilitation at Miners Hospital, director of the ICU, respiratory care services, medical care director of pulmonary rehab and medical director of the federal black lung clinic at Windber Medical Center. (CX 3 at p.5). Dr. Begley testified that it is his opinion, within a reasonable degree of medical certainty, that the miner was affected by coal workers' pneumoconiosis. (CX 3 at p. 13). He based this opinion on the miner's long history of exposure to coal dust, his recurrent abnormal x-rays, and the finding of coal workers' pneumoconiosis at

the time of his death. *Id.* Dr. Begley testified that in his clinical record review, there was evidence of pulmonary dysfunction and disability during the miner's life and cited pages five and six of Dr. Perper's November 27, 2002 report in support, which were referencing a 1987 exam. (CX 3 at pp. 15-16).

Dr. Begley testified that the miner's symptoms of dyspnea on exertion, abnormal pulmonary function studies, and abnormal arterial blood gas studies reflect the miner's pulmonary impairment and disability. (CX 3 at p. 16) He cited Dr. Schaaf's June 7, 2001 pulmonary function study as a basis for this opinion. (CX 3 at p. 18). Dr. Begley testified that it is his opinion, within a reasonable degree of medical certainty that the miner's pulmonary impairment would inhibit him from participating in his last employment in the coal mining industry. *Id.* He testified that Dr. Rizkalla's pathologic findings explain the miner' lifetime dysfunction and he considered the miner's smoking history to be mild to moderate and noted that the miner stopped smoking twenty years prior to death. (CX 3 at p. 19). Dr. Begley opined that coal dust exposure can cause centrilobular emphysema. (CX 3 at p. 20).

Dr. Begley testified that the miner's significant coal workers' pneumoconiosis was a substantial contributing factor in his demise. (CX 3 at p. 22). Dr. Begley testified that it is his understanding that coal workers' pneumoconiosis contributed to the miner's death because the miner had significant pulmonary impairment and significant arterial hypoxemia, which in the setting of fixed cardiac lesions worsened the ischemia. (CX 3 at pp. 22-23). Dr. Begley testified that it is his opinion the miner suffered from cor pulmonale. (CX 3 at p. 24). Dr. Begley testified, in reference to the miner's 1987 arterial blood gas study values, that the sum of the miner's PO2 and PCO2 were 112 and they should be 140, which is normal. (CX 3 at p. 30). He stated that this rule of 140, regardless of age, is pulmonary physiology. *Id*.

Dr. Begley testified that he did not know the miner had a cardiac arrest in the 1980's. (CX 3 at p. 39). Dr. Begley testified that he didn't review the records to look at the fact that the miner had a significant weight gain in the last several years of his life and that he did not see a report that indicated that the miner had fat in his cardiac muscle. (CX 3 at p. 40). Dr. Begley testified that the fact that the miner had a heart attack in the 1980's does not affect his opinion as to the mechanics of death, and Dr. Oesterling's opinion that there was fatty infiltrate in the left ventricle has no bearing on his conclusion. (CX 3 at p. 53-54).

Conclusions of Law

Miner's Claim

Benefits are provided to miners who are totally disabled due to pneumoconiosis arising out of coal mine employment. § 718.204(a). A miner shall be considered totally disabled if the irrebuttable presumption in § 718.304 applies. If that presumption does not apply, a miner shall be considered totally disabled if his pulmonary or respiratory impairment, standing alone, prevents him from performing his usual coal mine work and comparable and gainful work. § 718.204(b)(1). In the absence of contrary probative evidence, a miner's total disability shall be established by pulmonary function studies showing the values equal to or less than those in Appendix B, blood gas studies showing the values in Appendix C, the existence of cor

pulmonale with right sided congestive heart failure, or the reasoned and documented opinion of a physician finding that the miner's pulmonary or respiratory impairment prevented him from engaging in his usual coal mine work and comparable and gainful work. § 718.204(b)(2).

None of the evidence demonstrates that the miner had complicated pneumoconiosis. Therefore, the irrebuttable presumption at § 718.304 does not apply.

Of the six pulmonary function studies in the record, only the June 7, 2001 study produced qualifying values at rest; however, the values were non-qualifying after bronchodilators were administered. In addition, tracings in the record do not accompany the test, and the physician administering the test indicated that the results could not be reproduced, which raises the question of its validity. *See Estes v. Director, OWCP*, 7 B.L.R. 1-414 (1984). I find that Claimant has not established total disability with the pulmonary function study evidence.

There were four arterial blood gas studies in the record, none of which were qualifying. Therefore, I find that Claimant has not established total disability with the arterial blood gas study evidence.

There is evidence that the miner suffered from cor pulmonale in the autopsy report. In addition to the autopsy prosector, Dr. Perper, a reviewing pathologist, concurred that the slides revealed cor pulmonale. Two other reviewing pathologists, Drs. Bush and Oesterling opined that the miner did not have cor pulmonale. However, as set forth below, I found their opinions to be less credible on the subject than Dr. Rizkalla, the prosector, and Dr. Perper. Consequently, I find that Claimant has established the existence of cor pulmonale.

Drs. Fino, Schaaf, Begley, and Perper opined that the miner was totally disabled from performing his previous coal mine work or comparable coal mine employment. Alternatively, Drs. Sobieski, Malhotra, Ignacio, Castle and Rosenberg opined that the miner was not totally disabled. Dr. Klemens did not render an opinion about the miner's pulmonary capabilities. Dr. Fino was aware of the exertional requirements of the miner's job and he based his opinion on the objective studies and the miner's physical symptoms, notwithstanding the fact that the ventilatory studies and arterial blood gas studies were non-qualifying, and he acknowledged that the miner was too disabled to perform the heavy exertional requirements of his previous coal mine work. Similarly, Dr. Schaaf also discussed the demands of the miner's usual coal mine work and concluded that he would not be able to perform it based upon the objective tests and his complaints. Dr. Begley found the miner's objective studies to be abnormal and when considered with his physical complaints concluded that the miner would have been unable to perform his previous coal mine job. I find that these opinions are well-documented and well-reasoned and entitled to great weight.

Drs. Sobieski and Castle based their opinions predominantly on the non-qualifying studies, and Dr. Castle emphasized repeatedly that the lack of qualifying results was the key component in his opinion, notwithstanding the fact that the miner's studies, although non-qualifying, were also deemed abnormal by most of the reviewing physicians. Therefore, I find their opinions to be entitled to less weight than Drs. Fino, Schaaf, and Begley's opinions. Dr. Rosenberg acknowledged that the miner had airflow obstruction, but did not discuss the

finding in relation to the miner's coal mine work that required heavy exertion. In addition, Drs. Malhotra and Ignacio did not discuss the miner's pulmonary status in relation to his coal mine employment; therefore, I find that their opinions are entitled to less weight.

Of the pathologists, Drs. Bush and Oesterling, unlike Dr. Perper, did not discuss the heavy exertional requirements of the miner's job, and Dr. Bush opined that the miner's mild degree of simple coal workers' pneumoconiosis did not cause respiratory impairment or disability during his lifetime, which is contradicted by the later objective studies they reviewed. Again, while these studies were non-qualifying, almost all of the physicians agreed that they were not normal and demonstrated some impairment.

As the opinions of the physicians finding total disability are better reasoned and better supported by all of the evidence in the record, I find that Claimant has established that the miner was totally disabled pursuant to § 718.204(b)(2). Moreover, weighing all of the medical evidence together, including the pulmonary function studies, arterial blood gas studies, evidence of cor pulmonale, and the better reasoned medical opinions, I find that Claimant has established that the miner was totally disabled.

A miner shall be considered totally disabled due to pneumoconiosis if pneumoconiosis is a substantial contributor to the miner's disability. § 718.204(c). *Bonessa v. U.S. Steel Corp.*, 884 F.2d 726, 734 (3rd Cir. 1989). Drs. Schaaf, Perper, and Begley opined that the miner's total disability was due to pneumoconiosis. Dr. Sobieski opined that it is possible the miner had a disability because of coronary artery disease but needs further testing. Dr. Fino opined that the miner was disabled due to cardiac disease and smoking, but not to pneumoconiosis. Dr. Castle opined that it is possible that the miner is disabled due to his coronary artery disease, age, and deconditioning, which are not related to the inhalation of coal mine dust. Drs. Malhtotra, Ignacio, and Rosenberg opined that the miner was not totally disabled and Dr. Klemens did not discuss the issue at all.

Of these opinions, Drs. Malhotra, Ignacio, Rosenberg, and Klemens are entitled to little weight because they did not find the miner to be disabled or did not discuss the issue. In addition, Dr. Sobieski's opinion is equivocal and he initially opined that the miner did not have a pulmonary impairment. Therefore, his opinion is entitled to little weight. Dr. Fino acknowledged that the duration of the miner's coal mine employment was sufficient to explain his disability but stated that the "timeline" of events do not support it as a cause, without further explanation. Given that Dr. Fino acknowledged that the miner is disabled and that pneumoconiosis can be a progressive disease process, I find this explanation to be insufficient and I accord it less weight. Similarly, Dr. Castle never explained how he was able to exclude coal workers' pneumoconiosis as a contributor to the miner's disability.

While Dr. Begley opined that the miner was totally disabled, he attributed the miner's disability solely to his pneumoconiosis. Given the miner's significant cigarette smoking history, obesity, and severe coronary artery disease, and the fact that he testified that he was not aware that the miner had had an earlier heart attack, I find that his opinion is not well reasoned and is entitled to less weight.

Like Dr. Begley, Dr. Schaaf was aware of the miner's smoking history and cardiac status but did not explain how or why he eliminated them from consideration. I find that his opinion is also entitled to less weight.

Dr. Perper's opinion, however, best reflects all the evidence in the record. He considered both the miner's smoking and occupational histories, and cardiac status when rendering his opinions. Accordingly, I find that his opinion is entitled to great weight.

Weighing the medical opinion evidence together, I find that Claimant has established that the miner was totally disabled due to pneumoconiosis. As the onset date of his total disability is not clear, benefits will be paid as of October 1, 2000, the date the claim was filed. See § 725.503(b).

Survivor's Claim

For the purpose of adjudicating survivors' claims filed on or after January 1, 1982, death will be considered due to pneumoconiosis where medical evidence establishes that pneumoconiosis was the cause of the miner's death, where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death, or the death was caused by complications of pneumoconiosis, or where the presumption in § 718.304 is applicable. See § 718.205(c). Pneumoconiosis is a substantially contributing cause of death if it hastened the miner's death. *Lukosevicz v. Director, OWCP*, 888 F.2d 1001 (3rd Cir. 1989).

There is no evidence that pneumoconiosis was the direct cause of the miner's death; therefore, Claimant has not proven death due to pneumoconiosis pursuant to § 718.205(c)(1). There is no evidence that the miner suffered from complicated pneumoconiosis and therefore Claimant has not established death due to pneumoconiosis pursuant to § 718.205(c)(3).

The death certificate lists the immediate cause of death as acute myocardial infarction and longstanding coronary artery disease. A death certificate, in and of itself, is an unreliable report of the miner's condition. *Smith v. Camco Mining Inc.*, 13 B.L.R. 1017 (1989); *Addison v. Director, OWCP*, 11 B.L.R. 1-68 (1988).

It is not disputed that the miner suffered from simple coal workers' pneumoconiosis at the time of his death. Six physicians rendered opinions as to whether and to what extent pneumoconiosis contributed to or hastened the miner's death pursuant to § 718.205(c). Drs. Rizkalla, Perper, and Begley opined that the pneumoconiosis contributed to or hastened the miner's death. Drs. Bush, Oesterling, and Castle determined that pneumoconiosis did not contribute to or hasten the miner's death. The medical evidence in this case is extensive and complex. The crux of the matter is centered on whether the miner suffered from cor pulmonale, caused by his pneumoconiosis and emphysema, which then resulted in a fatal arrhythmia and death.

Dr. Rizkalla, the autopsy prosector, opined that the miner's severe atherosclerotic coronary artery disease and his moderately severe centrilobular emphysema associated with the coal workers' pneumoconiosis was a substantial contributing factor in the death. Dr. Rizkalla

explained that the right ventricle of the heart was dilated and thickened to twice the normal size and he had cor pulmonale. He explained that the miner's severe atherosclerotic heart disease narrowed the vessels and increased heart mass, thus reducing the quantity of blood coming through the arteries. He explained that the quality of the miner's blood was reduced from the miner's coal workers' pneumoconiosis and smoking, resulting in reduced oxygen saturation, which could precipitate a cardiac arrhythmia. Dr. Rizkalla opined that the finding of cor pulmonale was crucial in his attributing death to the pneumoconiosis and emphysema, and that it is unimaginable that the miner would have the amount of coal dust and macules in his lung that he did and not have cor pulmonale. Dr. Rizkalla also addressed the objective data in relation to the miner's physical condition during his lifetime and explained how he arrived at his opinion. I find that his opinion is well documented and well reasoned and accord it great weight.

Dr. Perper opined that coal workers' pneumoconiosis and the associated centrilobular emphysema was a substantial contributory cause of the miner's death, both directly and indirectly, through pulmonary insufficiency and through hypoxemia, triggering or aggravating an arrhythmia, on the background of marked heart disease. Dr. Perper based his diagnosis of cor pulmonale on the measurements of the right ventricle at autopsy, and that the intrapulmonary blood vessels showed thickening and sclerosing of the wall. This is also consistent with Dr. Rizkalla's findings. Dr. Perper based his opinion on the slides, gross description, and objective studies during the miner's lifetime, his occupational and social histories, and his physical complaints. I find that his opinion is also well documented and well reasoned and supported by all of the evidence in the record. I find that it is entitled to great weight.

Dr. Begley opined that the miner's coal workers' pneumoconiosis accelerated his demise by causing significant progressive arterial hypoxemia that worsened his cardiac ischemia and led to his ultimate demise. Dr. Begley, on the other hand, appeared unfamiliar with the miner's history. He was unaware the miner suffered a previous heart attack and appeared to downplay the miner's significant smoking history, which he described as mild to moderate. Dr. Begley also seemed unaware of the miner's obesity. Therefore, I find that his opinion is not well reasoned and I accord it less weight than the opinions of Drs. Rizkalla and Perper.

Dr. Bush opined that death was not caused by, contributed to, or hastened by any chronic dust disease arising from coal mine employment, and the degree of chronic dust disease related to coal mine employment is very limited and could not have been a factor in the events leading to death. Dr. Bush's medical report did not address cor pulmonale but he testified in his deposition that he did not discuss the findings because he did not see cor pulmonale himself. I do not find this to be credible. Cor pulmonale is a significant finding and doctors generally discuss the absence of findings as well as the presence of them in their medical reports. Moreover, Dr. Bush testified that cor pulmonale typically produces abnormal pulmonary function studies, which the miner did not have. Although the miner's pulmonary function studies were non-qualifying, almost all of the later reviewing physicians, including Dr. Fino, observed some abnormalities such as hypoxemia. Consequently, I find that Dr. Bush's opinion is entitled to little weight.

Dr. Oesterling opined that the miner was experiencing significant ischemic cardiomyopathy during his terminal days and weeks, which resulted in severe passive congestion with pulmonary edema, which could lead to cardiac arrhythmia, cardiac arrest and death.

Dr. Oesterling opined that the miner did not have cor pulmonale, but that his right ventricle was enlarged due to fatty deposits from his obesity. Dr. Rizkalla, however, agreed with Dr. Oesterling that the right ventricle can contain some fatty cells but stated that the fat infiltration will not increase the thickness up to eight millimeters, as in the miner's case. Dr. Rizkalla further explained that the presence of fat in the right ventricle does not mean that a person does not have cor pulmonale. Dr. Rizkalla also explained that he disagreed with Dr. Oesterling's observation of a recent cardiac event before death and stated that Dr. Oesterling was likely describing the presence of collagen, which indicates an old process. I find that Dr. Oesterling's opinion is well documented and well reasoned but not as well reasoned as those of Drs. Rizkalla and Perper.

Dr. Castle opined that the miner experienced a sudden cardiac event and there is no evidence that hypoxemia played a role in precipitating an arrhythmia, that the miner's death was due to cardiac disease and was unrelated to any lung disease including coal workers' pneumoconiosis, and the miner would have died as and when he did regardless of coal workers' pneumoconiosis. I find Dr. Castle's opinion not to be supported by the medical evidence in the record. Dr. Castle did not discuss the presence of cor pulmonale and any effect it may have had on the miner's cardiac function and death. Dr. Castle also did not believe that the miner had any hypoxemia at the time of his death, which is contrary to what other reviewers noted, including Dr. Fino.

In terms of the qualifications of the pathologists rendering opinions, I find that Dr. Perper is the most qualified with respect to experience, academic appointments, and published research. I find Drs. Rizkalla and Oesterling to be equally qualified, and I find Dr. Bush to be the least qualified, as he is retired and currently only performing consultations.

I find that not only is Dr. Perper the most qualified physician, I also find his reports and testimony, along with Dr. Rizkalla's, to be better reasoned, more credible, and better supported by all the evidence in the record. I find that their opinions - that the miner had cor pulmonale, that his hypoxia from his coal workers' pneumoconiosis and centrilobular emphysema, combined with his weakened condition due to his atherosclerotic heart disease, caused an arrhythmia resulting in sudden death - best synthesizes all of the medical evidence of record. Weighing all of the evidence together, I find that the claimant has met her burden of showing that the miner's pneumoconiosis hastened his death and was a substantially contributing cause of the miner's death under § 718.205(c).

As Claimant established all elements of entitlement, I find that she is entitled to benefits under the Act. Benefits will be awarded as of February 1, 2002, the month in which the miner died.

Thirty days are hereby allowed to Claimant's counsel for the submission of an application for attorney's fees. His attention is directed to §§ 725.365 and 725.366 of the Regulations. A service sheet showing service upon all parties, including the claimant, must accompany the application. Parties have twenty days following receipt of such application within which to file any objections.

ORDER

IT IS ORDERED THAT Island Creek Coal Co.:

1. Pay Claimant benefits as of October 1, 2000, augmented by one dependent; and

2. Pay Claimant benefits as of February 1, 2002, with no augmentation.

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DANIEL L. LELAND
Administrative Law Judge

NOTICE OF APPEAL RIGHTS. Pursuant to 20 C.F.R. Section 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date this Decision and Order was filed in the Office of the District Director, by filing a notice of appeal with the *Benefits Review Board at P.O. Box 37601, Washington, D.C. 20013-7601*. A copy of a notice of appeal must also be served on Donald S. Shire, Esq., Associate Solicitor for Black Lung Benefits. His address is Frances Perkins Building, Room N-2117, 200 Constitution Avenue, N.W., Washington, D.C. 20210.